Amendments to the Claims:

This listing of claims will replace all prior versions and listings of claims in the application:

Listing of Claims:

1. (Currently amended) A method to aid in predicting susceptibility of a mammalian subject to development or growth of a steroid hormone responsive cancer in a mucosal epithelial tissue, the method comprising:

(a) quantitating and/or detecting an immunoglobulin inhibitor of steroid hormone responsive cell growth in [a] at least one specimen of body fluid or secretion obtained from said subject, wherein said inhibition of steroid hormone responsive cell growth is capable of being reversed by ~~binding a~~ said steroid hormone ~~to a cellular steroid hormone receptor that is active for promoting cell growth~~[,];

(b) determining whether said immunoglobulin inhibitor is absent or deficient in said at least one body fluid or secretion; and

(c) using the determination from step (b), determining that said ~~an~~ absence or deficiency of said immunoglobulin inhibitor ~~suggesting or indicating~~ indicates that a steroid hormone responsive mucosal epithelial tissue in said subject is contacted by insufficient immunoglobulin inhibitor to inhibit steroid hormone responsive cell growth in said tissue ~~secretes or is bathed by less than a cell growth inhibitory amount of said immunoglobulin inhibitor~~.

2. (Currently amended) The method of claim 1 further comprising obtaining a sample of at least one body fluid or secretion chosen from the group consisting of ~~serum, plasma,~~ colostrum, breast aspirates, saliva, tears, bronchial secretions, nasal mucosa, prostatic fluid, urine, semen or seminal fluid, vaginal secretions, ovarian aspirates, stool, and mucous secretions from the small intestine or stomach.

3. (Previously presented) The method of claim 1 wherein said quantitating and/or detecting comprises measuring the amount and/or activity of said immunoglobulin inhibitor in a specimen of body fluid or secretion from said subject.

4. (Currently amended) The method of claim 1 wherein said quantitating and/or detecting comprises ~~substantially~~ depleting steroid hormone from said specimen of body fluid or secretion to yield a steroid hormone depleted specimen, and assaying said steroid hormone depleted specimen for steroid hormone reversible inhibition of steroid hormone responsive cancer cell proliferation.

5. (Currently amended) The method of claim 4 wherein said assaying comprises:

maintaining a population of steroid hormone-responsive cancer cells in a nutrient medium containing calcium ion and substantially no free ferric ion, said cells also being steroid hormone responsive for *in vivo* proliferation if implanted in a suitable host;

adding steroid hormone to said medium sufficient to stimulate cell growth under cell growth promoting culture conditions;

adding a steroid hormone free specimen of a body fluid or secretion to said medium, to yield a test mixture;

incubating said test mixture under cell growth promoting conditions;

after said incubating, measuring the cell population in said test mixture;

measuring the cell population in a control incubation mixture like said test mixture, except lacking said specimen;

optionally, testing said specimen for cytotoxic effects on said cells;

measuring the differences in cell number between said cell populations before and after said incubation, a significant increase in ~~said~~ cell population doublings indicating the absence of inhibition of cell growth by said specimen in the presence of said steroid hormone, and a significant lack of increase in ~~said~~ cell population doublings not attributable to cytotoxic effects of said specimen indicating inhibition of cell growth by said specimen in the presence of said steroid hormone.

6. (Currently amended) The method of claim ~~5~~ 1 wherein said inhibition of steroid hormone responsive cell growth is capable of being reversed by ~~adding of steroid hormone to said medium comprises adding~~ an amount of steroid hormone that is in the physiological concentration range for said steroid hormone in said mammal.

7. (Currently amended) An *in vitro* method of detecting loss of immunoglobulin regulation of steroid hormone responsive cell growth of a mucosal epithelial cell comprising:

(a) assaying for the presence of a poly-Ig receptor on said mucosal epithelial cell;

(b) optionally, testing said poly-Ig receptor for ability to bind dimeric/polymeric IgA or polymeric IgM;

(c) using assay results from step (a) and, optionally, test results from step (b), determining that an absence or deficiency of said receptor indicates loss of immunoglobulin regulation of growth of said cell, wherein immunoglobulin regulation comprises steroid hormone reversible inhibition of steroid

hormone responsive mucosal epithelial cell growth by dimeric/polymeric IgA or polymeric IgM ~~determining that a poly-Ig receptor in a non-cancerous mucosal epithelial cell has the property of being able to mediate inhibition by IgA or IgM of steroid hormone responsive cell growth, wherein said inhibition of steroid hormone responsive cell growth is reversible by binding said steroid hormone to a cellular steroid hormone receptor that is active for promoting cell growth; and~~

~~assaying for inability of a mucosal epithelial cell to bind IgA or IgM by other than antibody-antigen recognition based association~~.

8. (Currently amended) A method of detecting a mediator of immunoglobulin inhibition of steroid hormone responsive cell growth wherein said inhibition can be reversed by ~~binding a~~ said steroid hormone ~~to a cellular steroid hormone receptor that is active for promoting cell growth~~, the method comprising:

(a) detecting a poly-Ig receptor in a mucosal epithelial cell;

(b) optionally, testing said poly-Ig receptor for *in vitro* activity ~~formediating~~ for mediating said steroid hormone reversible immunoglobulin inhibition of steroid hormone responsive cell growth; and

(c) using the detection from step (a) and, optionally, test results from step (b), determining that said poly-Ig receptor is capable of mediating immunoglobulin inhibition of steroid hormone responsive cell growth wherein said inhibition is capable of being reversed by ~~binding~~ said steroid hormone ~~to a cellular steroid hormone receptor that is active for promoting cell growth~~.

9. (Currently amended) A method of detecting a gene coding for a mediator of immunoglobulin inhibition of steroid hormone responsive cell growth comprising:

detecting the presence of a poly-Ig receptor gene in a mucosal epithelial cell; and

determining whether [a] the poly-Ig receptor encoded by said gene has the property of being capable of mediating steroid hormone reversible ~~immunoglobulin~~ inhibition by dimeric/polymeric IgA or polymeric IgM of steroid hormone responsive cell growth wherein said inhibition is reversed by ~~binding a~~ said steroid hormone ~~to a cellular steroid hormone receptor that is active for promoting cell growth~~.

10. (Currently amended) A method of detecting a ~~genetic defect in~~ variant poly-Ig receptor gene coding for a ~~mediator of immunoglobulin inhibition of steroid hormone responsive cell growth~~ protein that lacks or has reduced ability to mediate inhibition by dimeric/polymeric IgA or polymeric IgM of

steroid hormone responsive cell growth in a mucosal epithelial cell compared to the native poly-Ig receptor, the method comprising:

identifying a loss of heterozygosity or an allelic imbalance in a poly-Ig receptor gene; or

carrying out site directed mutagenesis in a non-cancer tissue culture cell model whereby a domain of a poly-Ig receptor gene is altered,

identifying at least one said altered domain in said poly-Ig receptor gene that causes loss of ability of said poly-Ig receptor to mediate inhibition of steroid hormone responsive cell growth by an immunoglobulin inhibitor, wherein said inhibition is capable of being reversed by ~~binding a~~ said steroid hormone ~~to a cellular steroid hormone receptor that is active for promoting cell growth~~;

screening a genomic or cDNA library of a cancerous mucosal epithelial cell for a poly-Ig receptor gene; and

matching said screened poly-Ig receptor gene from said cancerous cell to a poly-Ig receptor gene from said non-cancerous cell culture model which contains an altered domain that is correlated to loss of inhibition mediating ability, whereby a ~~genetically defective~~variant poly-Ig receptor gene is identified which encodes a protein that is incapable or less capable of mediating inhibition by dimeric/polymeric IgA or polymeric IgM of steroid hormone responsive cell growth in a mucosal epithelial cell compared to the native poly-Ig receptor.

11. (Currently amended) A method of detecting expression of a defective mediator of immunoglobulin inhibition of steroid hormone responsive cell growth in cells from a specimen of mucosal epithelial tissue, the method comprising:

expressing in said cells the protein encoded by the variant poly-Ig receptor gene detected ~~detecting a poly-Ig receptor protein in said specimen which is encoded by the genetically defective poly-Ig receptor gene identified~~ according to the method of claim 10, to provide an expressed variant poly-Ig receptor protein; and

testing said variant protein for ability to mediate steroid hormone reversible inhibition of steroid hormone responsive cell growth in said mucosal epithelial cells.

12. (Currently amended) A method to aid in predicting susceptibility of a mammalian subject to development of breast cancer comprising:

(a) detecting the loss or impairment of negative regulation of breast tissue proliferation by the secretory immune system in said subject, said detecting comprising testing for loss or reduction of

immunoglobulin inhibition of steroid hormone responsive cell growth in said tissue by dimeric/polymeric IgA, polymeric IgM or IgG1[,]; and

(b) using the detection from step (a) to predict susceptibility of said subject to development of breast cancer wherein ~~said inhibition is capable of being reversed by binding a steroid hormone to a cellular steroid hormone receptor that is active for promoting cell growth~~ a detected loss or impairment of said inhibition indicates greater risk of development of breast cancer.

13. (Currently amended) A method to aid in predicting increased susceptibility of a mammalian subject to development or growth of a steroid hormone responsive cancer in a mucosal epithelial tissue, the method comprising:

(a) assaying a specimen of mucosal epithelial tissue obtained from said subject for the presence of a poly-Ig receptor; ~~and~~

(b) optionally, testing to determine~~determining~~ whether said poly-Ig receptor is capable of mediating immunoglobulin inhibition of steroid hormone responsive cell growth, wherein said inhibition is capable of being reversed by ~~binding a~~ said steroid hormone ~~to a cellular steroid hormone receptor that is active for promoting cell growth,~~;

(c) using assay results from step (a) and, optionally, test results from step (b), determining increased susceptibility of said subject to development or growth of a steroid hormone responsive cancer in said mucosal epithelial tissue, wherein an absence of said receptor or an absence of activity of said receptor for mediating said immunoglobulin inhibition ~~suggesting~~ indicates that said tissue lacks sufficient functional mediators of immunoglobulin inhibition to deter development or growth of a steroid hormone responsive cancer in said mucosal epithelial tissue.

14. (Currently amended) A method to aid in detecting transformation of a mucosal epithelial cell from normally steroid hormone responsive to a steroid hormone responsive cancerous condition, the method comprising:

~~determining that a Fcγ receptor has the property of being able to mediate inhibition by IgG1 or IgG2 of steroid hormone responsive cell growth, wherein said inhibition is capable of being reversed by binding a steroid hormone to a cellular steroid hormone receptor that is active for promoting cell growth;~~

~~optionally, testing said cells for presence of a Fcγ receptor; and~~

(a) assaying a population of said mucosal epithelial cells for loss of said receptor or inactivity of said receptor for binding IgG1 or IgG2~~, wherein inactivity for binding IgG1 or IgG2 is suggestive of said transformation~~;

(b) optionally, testing said cells for presence of a Fcγ receptor; and

(c) using assay results from step (a), and, optionally, test results from step (b) to determine that said mucosal epithelial cells have transformed from a normally steroid hormone responsive condition to a steroid hormone responsive cancerous condition, wherein absence of said Fcγ receptor or inactivity of said Fcγ receptor for binding IgG1 or IgG2 is indicative of said transformation.

15. (Currently amended) A method to aid in detecting progression of a steroid hormone responsive malignant mucosal epithelial cell to an autonomous cancer cell, the method comprising:

~~determining that a poly-Ig receptor has the property of being able to mediate inhibition by IgA or IgM of steroid hormone responsive cell growth, wherein said inhibition is capable of being reversed by binding a steroid hormone to a cellular steroid hormone receptor that is active for promoting cell growth;~~

(a) optionally, testing ~~said cells~~ an autonomous cancer cell for presence of a poly-Ig receptor;

(b) ~~testing~~ assaying said autonomous cancer cell for ability to bind dimeric/polymeric IgA or polymeric IgM ~~by other than antibody antigen recognition based association~~;

(c) using assay results from step (b), and, optionally, test results from step (a) to detect the absence of said poly-Ig receptor and/or the absence of ability of said autonomous cancer cell to bind dimeric/polymeric IgA or polymeric IgM, wherein said absence or loss indicates progression of a steroid hormone responsive malignant mucosal epithelial cell to an autonomous cancer cell.

16. (Canceled)

17. (Currently amended) A method to aid in detecting or diagnosing cancer in a mammalian subject comprising determining, in a population of cells taken from a mucosal epithelial tissue specimen obtained from said subject, at least one of a first set of conditions selected from the following:

absence or diminution of immunoglobulin inhibition of steroid hormone responsive cell growth, wherein said inhibition is capable of being reversed by ~~binding a~~ said steroid hormone ~~to a cellular steroid hormone receptor that is active for promoting cell growth~~;

absence or diminution of at least one immunoglobulin inhibitor of steroid hormone responsive cell growth from a body fluid or secretion secreted by or bathing said tissue, wherein inhibition by said inhibitor is capable of being reversed by ~~binding a~~ said steroid hormone ~~to a cellular steroid hormone receptor that is active for promoting cell growth~~,

absence or diminution of a poly-Ig receptor in said cells,

absence of a poly-Ig receptor gene from said cells,

absence of heterozygosity for said poly-Ig receptor gene in said cells,

absence or diminution of a Fcγ receptor in said cells,

absence of a Fcγ receptor gene from said cells,

absence of heterozygosity for said Fcγ receptor gene in said cells,

and, optionally, detecting at least one of a second set of conditions selected from the following:

absence or diminution of TGFβ regulation of cell growth,

absence or diminution of a TGFβ receptor in said cells,

absence of a TGFβ receptor gene from said cells,

absence of heterozygosity for said TGFβ receptor gene in said cells, and

using the results of said determinations and detections to aid in detecting or diagnosing cancer in a mammalian subject, said absence or diminution ~~being suggestive or indicative of~~indicating the presence of a cancerous or precancerous lesion in said patient, and an absence of one or more of said conditions ~~being suggestive or indicative of~~indicating the absence of a cancerous or precancerous lesion in said patient.

18. (Currently amended) A method to aid in staging a cancer of a mucosal epithelial tissue comprising:

determining, in a specimen of neoplastic cells obtained from said cancer, whether said cells are stimulated by a steroid hormone to proliferate in ~~a suitable cell growth nutrient medium~~an *in vitro* cell proliferation assay;

if it is determined that said cells are stimulated by said steroid hormone to proliferate, determining the amount of immunoglobulin inhibitor in a specimen of body fluid or secretion secreted by or bathing said mucosal epithelial tissue which is in a form that is active for inhibiting steroid hormone responsive cell proliferation, wherein said inhibition ~~by said immunoglobulin inhibitor~~ is capable of being reversed by ~~binding a~~ said steroid hormone ~~to a cellular steroid hormone receptor that is active for promoting cell growth;~~ determining at least one of the following conditions:

loss or diminution of a TGFβ receptor in said cells,

loss of a TGFβ receptor gene in said cells,

loss of heterozygosity for said TGFβ receptor gene in said cells,

loss or diminution of a poly-Ig receptor in said cells,

loss of a poly-Ig receptor gene in said cells,

loss of heterozygosity for said poly-Ig receptor gene in said cells,

loss or diminution of a Fcγ receptor in said cells,

loss of a Fcγ receptor gene in said cells,

loss of heterozygosity for said Fcγ receptor gene in said cells; and

using said determinations to aid in staging said cancer of a mucosal epithelial tissue wherein one or more said loss or diminution indicates a more advanced stage.

19. (Currently amended) A method to aid in prognosis of a mammalian cancer patient comprising:

a) obtaining from said patient a specimen of body fluid, a secretion secreted by or bathing a mucosal epithelial tissue, obtaining from said patient a specimen of ~~or~~ neoplastic cells from a mucosal epithelial tissue;

b) in said specimen of body fluid or secretion, determining the lack of a cell growth inhibitory amount of at least one immunoglobulin inhibitor of steroid hormone responsive cell growth, wherein inhibition by said immunoglobulin inhibitor is capable of being reversed by ~~binding a~~ said steroid hormone ~~to a cellular steroid hormone receptor that is active for promoting cell growth~~, and

c) additionally determining at least one of the following conditions:

c-1) in said specimen of neoplastic cells from said tissue, the loss or diminution of a TGFβ receptor,

c-2) in said specimen of neoplastic cells from said tissue, the loss of a TGFβ receptor gene,

c-3) in said specimen of neoplastic cells from said tissue, the loss of heterozygosity for said TGFβ receptor gene,

c-4) in said specimen of neoplastic cells from said tissue, the loss or diminution of a poly-Ig receptor,

c-5) in said specimen of neoplastic cells from said tissue, the loss of a poly-Ig receptor gene,

c-6) in said specimen of neoplastic cells from said tissue, the loss of heterozygosity for said poly-Ig receptor gene,

c-7) in said specimen of neoplastic cells from said tissue, the loss or diminution of a Fcγ receptor,

c-8) in said specimen of neoplastic cells from said tissue, loss of a Fcγ receptor gene,

c-9) in said specimen of neoplastic cells from said tissue, loss of heterozygosity for said Fcγ receptor gene; and

d) using said determinations from b) and c) to aid in prognosis of said patient, wherein the presence of one or more of said conditions ~~being~~ is ~~suggestive of~~ indicative of at least some degree of

reduced prognosis of said patient, and an absence of one or more of said conditions ~~being~~ is ~~suggestive or~~ indicative of at least some degree of favorable prognosis.

20. (Currently amended) A method to aid in treating cancer of a mucosal/epithelial tissue comprising

a) detecting in a population of cancer cells obtained from said tissue the presence of ~~ERγ~~ a high-affinity estrogen binding activity having a greater E_2 binding affinity than that of ERα or ERβ, and

b) using the detection from a), determining that the presence of said high-affinity estrogen binding activity indicates that said cancer cell is estrogen dependent for growth, or determining that the absence of said high-affinity estrogen binding activity indicates that said cancer cell is not estrogen dependent for growth.

21-65. (Canceled)

66. (Currently amended) The method of claim 5 wherein said significant increase or significant lack of increase in said cell population doublings is determined using the student's *t* test and wherein a value of $p < 0.05$ is significant.

67. (Currently amended) The method of claim 8 comprising testing said poly-Ig receptor for activity for mediating steroid hormone reversible inhibition by dimeric/polymeric IgA or polymeric IgM of steroid hormone responsive cell growth.

68. (Currently amended) A method of detecting a mediator of immunoglobulin inhibition of steroid hormone responsive cell growth wherein said inhibition can be reversed by ~~binding a~~ said steroid hormone ~~to a cellular steroid hormone receptor that is active for promoting cell growth~~, the method comprising:

(a) detecting an Fcγ receptor in a mucosal epithelial cell;

(b) optionally, testing said Fcγ receptor for *in vitro* activity for mediating said steroid hormone reversible immunoglobulin inhibition of steroid hormone responsive cell growth~~,~~; and

(c) using the detection from step (a) and, optionally, test results from step (b), to determine ~~determining~~ that said Fcγ receptor is capable of mediating immunoglobulin inhibition of steroid hormone responsive cell growth wherein said inhibition is capable of being reversed by ~~binding a~~ said ~~steroid hormone to a cellular steroid hormone receptor that is active for promoting cell growth~~.

69. (Currently amended) A method of detecting expression of a defective mediator of immunoglobulin inhibition of steroid hormone responsive cell growth in cells from a specimen of mucosal epithelial tissue, the method comprising:

expressing in said cells the protein encoded by the variant Fcγ receptor gene detected ~~detecting a Fcγ receptor protein in said specimen which is encoded by the genetically defective Fcγ receptor gene identified~~ according to the method of claim 80, to provide an expressed variant Fcγ receptor protein; and

testing said variant protein for ability to mediate steroid hormone reversible inhibition of steroid hormone responsive cell growth in said mucosal epithelial cells.

70. (Currently amended) The method of claim 1 wherein said immunoglobulin inhibitor comprises dimeric/polymeric IgA, polymeric IgM, IgG1 or IgG2, or any combination thereof.

71. (Currently amended) An *in vitro* method of detecting loss of immunoglobulin regulation of steroid hormone responsive cell growth of a mucosal epithelial cell comprising:

(a) assaying for the presence of an Fcγ receptor on a mucosal epithelial cell;

(b) optionally, testing said Fcγ receptor for ability to bind IgG1 or IgG2; and

(c) using assay results from step (a) and, optionally, test results from step (b), determining that an absence or deficiency of said receptor indicates loss of immunoglobulin regulation of growth of said cell, wherein immunoglobulin regulation comprises steroid hormone reversible inhibition of steroid hormone responsive mucosal epithelial cell growth by IgG1 or IgG2 ~~determining that an Fcγ receptor in a non-cancerous mucosal epithelial cell has the property of being able to mediate immunoglobulin inhibition of steroid hormone responsive cell growth, wherein said inhibition of steroid hormone responsive cell growth is capable of being reversed by binding said steroid hormone to a cellular steroid hormone receptor that is active for promoting cell growth; and~~

~~assaying for inability of a mucosal epithelial cell to bind IgG1 or IgG2 by other than antibody-antigen recognition based association~~.

72. (Currently amended) A method of detecting a gene coding for a mediator of immunoglobulin inhibition of steroid hormone responsive cell growth comprising:

detecting the presence of a Fcγ receptor gene in a mucosal epithelial cell; and

determining whether [a]the Fcγ receptor encoded by said gene has the property of being capable of mediating ~~immunoglobulin~~ inhibition by IgG1 or IgG2 of steroid hormone responsive cell growth, wherein said inhibition is reversible by ~~binding a~~ steroid hormone ~~to a cellular steroid hormone receptor that is active for promoting cell growth~~.

73. (Previously presented) The method of claim 1 further comprising detecting a poly-Ig receptor in said mucosal epithelial cell.

74. (Previously presented) The method of claim 1 further comprising detecting a Fcγ receptor in said mucosal epithelial cell.

75. (Currently amended) The method of claim 74 further comprising assessing the activity of said Fcγ receptor for mediating inhibition by IgG1 or IgG2 of steroid hormone responsive cell growth in a ~~suitable~~ an *in vitro* cell ~~culture~~ proliferation assay, wherein said inhibition is capable of being reversed ~~by binding a~~ said steroid hormone ~~to a cellular steroid hormone receptor that is active for promoting cell growth~~.

76. (Previously presented) The method of claim 1 comprising identifying an age range in said mammalian subject of increased susceptibility to developing breast cancer after exposure to a carcinogen.

77. (Previously presented) The method of claim 20 comprising increasing the number of B immunocytes in said mucosal/epithelial tissue producing IgA or IgM.

78. (Currently amended) The method of claim 20 comprising identifying an antagonist of ~~ERγ~~ said high-affinity estrogen binding activity.

79. (Previously presented) The method of claim 78 wherein said antagonist comprises tamoxifen.

80. (Currently amended) A method of detecting a ~~genetic defect in a~~ variant Fcγ receptor gene coding for a ~~mediator of immunoglobulin inhibition of steroid hormone responsive cell growth~~ protein that lacks or has reduced ability to mediate inhibition by IgG1 of steroid hormone responsive cell growth in a mucosal epithelial cell compared to said Fcγ receptor, the method comprising:

identifying a loss of heterozygosity or an allelic imbalance in a Fcγ receptor gene; or

carrying out site directed mutagenesis in a non-cancer tissue culture cell model whereby a domain of a Fcγ receptor gene is altered,

identifying at least one said altered domain in said poly-Ig receptor gene that causes loss of ability of said Fcγ receptor to mediate inhibition of steroid hormone responsive cell growth by an immunoglobulin inhibitor, wherein said inhibition is capable of being reversed by ~~binding a~~ said steroid hormone ~~to a cellular steroid hormone receptor that is active for promoting cell growth~~;

screening a genomic or cDNA library of a cancerous mucosal epithelial cell for a Fcγ receptor gene; and

matching said screened Fcγ receptor gene from said cancerous cell to a Fcγ receptor gene from said non-cancerous cell culture model which contains an altered domain that is correlated to loss of inhibition mediating ability, whereby a ~~genetically defective~~variant Fcγ receptor gene is identified which encodes a protein that is incapable or less capable of mediating inhibition by dimeric/polymeric IgA or polymeric IgM of steroid hormone responsive cell growth in a mucosal epithelial cell.

81. (New) The method of claim 1 wherein said steroid hormone is an estrogen or an androgen.

82. (New) A method to aid in predicting susceptibility of a mammalian subject to development of breast cancer comprising:

(a) identifying an age range in said subject during which DNA synthesis in breast tissue is expected to be increased compared to that of another age range;

(b) analyzing the IgA and/or IgM in blood and/or breast secretion of mature adult females;

(c) at an age within said age range of expected increased DNA synthesis, analyzing the IgA and/or IgM in blood and/or breast secretion of said subject;

(d) optionally, increasing the amount of IgA and/or IgM in the breast tissue of said subject;

(e) comparing the IgA and/or IgM analysis of said subject, from step (c) to the IgA and/or IgM analysis of said mature adult females, from step (b), to yield comparative results; and

(f) using the comparative results from step (e), predicting the susceptibility of said subject to development of breast cancer wherein a comparatively lower level of dimeric/polymeric IgA and/or polymeric IgM in said blood or breast secretion indicates greater risk of said subject to development of breast cancer.

83. (New) The method of claim 82 wherein said comparatively lower level of dimeric/polymeric IgA and/or polymeric IgM in said blood or breast secretion indicates increased susceptibility to mutagenesis and/or carcinogenesis.

84. (New) The method of claim 82 comprising increasing the number of IgA- and/or IgM-producing immunocytes in the breast tissue of said subject during the age range identified in step (a).

85. (New) The method of claim 84 wherein increasing the number of said immunocytes comprises orally immunizing said subject.

86. (New) The method of claim 17 wherein inhibition by said at least one immunoglobulin inhibitor is reversible by a physiologic concentration of said steroid hormone.

87. (New) The method of claim 20 wherein said greater E_2 binding affinity is $\leq 1 \times 10^{-12}$ M.

88. (New) The method of claim 68 wherein said inhibition is reversible by a physiological concentration of said steroid hormone.

89. (New) The method of claim 1 comprising obtaining a serum or plasma sample from said subject.

90. (New) A method of treating cancer of a mucosal epithelial tissue in an individual in need thereof comprising:

a) detecting the presence or absence of high-affinity estrogen binding activity having greater E_2 binding affinity than that of ERα or ERβ in said tissue, according to the method of claim 20; and

b) contacting said tissue with an inhibitor of said high-affinity estrogen binding activity.